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THE UNIVERSITY OF TORONTO MEDICAL JOURNAL

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EDITORIAL.

What is an editorial—has it like the modes and manners changed with the years? In older times they were expressions of definite opinions, attempts to protect the gullible reader against the wiles and chicaneries of the opposite party. Of late they have become but mild statements of facts, from which the reader is left to digest and draw his own conclusions.

What are our aims in an editorial—other than to fill blank space—to educate, to coerce, to tyrannize or to amuse? The former is probably better left to abler hands—the latter more to our liking is out of place in these pages. Two are but left—hence we have attempted to vitriolic denunciation (the view point is ours) to stimulate the lower years to literary activity, and at the same time to divulge the sources of knowledge which they so glibly quote. We have little hope of seeing this occur during our time—but the ceaseless repetition of this theme may in time achieve its ends.

At any rate each editor owes the junior years a vote of thanks for the annual presentation of a subject for an editorial.

The Lower Years and the Medical Journal.

There has been always noticed an apparent indifference in shyness in these years when the subject of articles

for the Journal has been brought up. Whether they feel they are incompetent to deal with a subject which has clinical aspects or whether they feel that a non-clinical subject would have little interest to the remaining readers —is a moot question. However, in spite of this there remains the fact that we do not, are not, seldom have and if no radical change takes place this year, probably will not receive articles.

Who is better qualified to deal with an article in the basic sciences—one in whose memory the subject is fresh in all its finer details, or one who has forgotten all but the salient features? One can not write a successful article without a general background of the subject and its allied branches even though they do not materially appear in the paper.

In an attempt to bring this home we shall analyze last year's issues. Dividing the articles into years we find:

1—Staff present 18.
2—VI. year, 14.
3—V. year, 16.
4—IV. year, 3.
5—II. year, 1.
6—Reprints, 4.
7—An occasional contribution of some former student or faculty member.

The figures given are fairly accurate—in studying them from another aspect we find that probably the majority of the undergraduate articles are papers presented in the VI. year and at A.O.A. meetings.

The cause is apparently unknown, if by chance the junior years do not like the articles suggested to their year representatives then let them make their own suggestions. They more than a senior know what they best need and by doing so would fulfil a double duty —first obtaining articles which would be of the greatest value in their studies —secondly, of obtaining the value of research in literature necessary to publish a paper.

Biographies.

Recently we have heard some of the students criticizing the papers presented because of the lack of references. To one who is interested in the subject it may be a bar to further search of knowledge. If the paper is used in the preparation of another, the sources of information should be easily obtained, in them might be facts the author is seeking. It is a guide to original researches and monographs on the subject — which would save him much arduous work in the files or the cumulative quarterlies. It is but the labour of a few minutes which greatly enhances the value of the article to all who read it.

Eye Injuries.

By W. H. LOWRY, M.D., C.M.
Professor of Ophthalmology.

The Senior student will soon be out in practice on his own, and will be accepting the responsibilities of caring for various sorts of injuries and diseases, which tax his older and more experienced colleague not a little. Unfortunately the undergraduate cannot be taught in a crowded course, all the little things that only experience in practice can teach, and the young practitioner experiences many bumps and worries in the first few years, that are almost unavoidable.

The young doctor will be sure to encounter eye injuries very early in his practice. The garage and the factory expose workmen to injuries from flying fragments of metal, from emery wheels, and fast moving machinery, and perhaps the most common source of injury, is the small piece which flies from the "burred" chisel when hit by a hammer.

The patient is brought to the doctor with a history of having been hit with something which is going so fast, or is so small that he does not know just what it is. He has had some initial pain which may soon pass off, and he is not particularly uncomfortable, but his vision is blurred in the injured eye. Examination of the eye may show various appearances.

(1) The anterior chamber may be filled with blood.
(2) The anterior chamber may be empty and the iris resting closely to the posterior surface of the cornea.
(3) The pupil may be drawn off to one side, and a brownish nodule seen at the edge of the cornea.
(4) The anterior chamber may be undisturbed, but one may see a faint wound in the cornea and a hole in the iris in line with it.

Anyone of these appearances indicate a serious injury to the eye and the doctor must be careful to recognize his responsibility to the patient and to himself. To the patient, because proper advice and treatment minimizes the effect of the injury to the injured eye and spares the terrible consequences of a possible sympathetic inflammation in the good eye. To himself, because from advice and treatment may lead to such serious consequences, that he may be exposed to a suit for mal-practice.

In Ontario, Quebec, and other provinces, the Workmen's Compensation Board undertake to give the workman good treatment, and the doctor must either give good treatment or see that the Workman is directed to a centre where facilities for proper treatment can be obtained. The Board pays for transportation to, and from the centre, so that there is no excuse for the treatment not being obtained, unless the doctor fails to do his part, which is to direct his patient with a letter to the Board.

Having recognized the injury as a serious one, the practitioner should consider carefully the conditions observed in the injured eye.

In (1) the eye has probably been struck by a large foreign body, and the blow has caused bleeding from the iris ciliary body or choroid into the anterior chamber, or vitrious or both. The treatment should be rest, cold compresses and atropine 1%, 3 times a day, for a few days. In a few days the blood will become absorbed, and in favorable cases the vision will shortly improve to normal, or nearly normal. It is well, however, to give a guarded prognosis for the vision might be impaired by the formation of a traumatic Cataract, a dislocated lens, a detachment of the retina, a rupture of the choroid, or an

injury to the macula, which conditions are not readily observed until sometime after the injury.

In (2) The Cornea has been penetrated, the aqueous has escaped and the eye is soft. The treatment should be atropine 1% at once, bandage the eye snugly, and put the patient at rest for a few hours. By this time the anterior chamber will have reformed and we can examine the eye more carefully, to see whether the eye has been penetrated by a body that has been withdrawn, or whether by a body which has entered the eye. A localizing X-Ray should be taken to clear up this point.

In (3) the Cornea has been penetrated and the iris has prolapsed into and through the wound, the doctor should cocainize the eye and if necessary use a general anaesthetic, flush out the conjunctival sac with boracic solution, and carefully cut off the prolapsed portion of the iris, and free it from the edges of the wound. In doing this he must be careful not to touch an uninjured lens with his instrument for fear of causing a traumatic Cataract. Plenty of Bichloride ointment 1-3000 should be placed between the eye lid and the eye, along with 1% atropine, and the eye snugly bandaged, and rest in bed being insisted upon for a few days.

As before an X-Ray should be taken to see whether a foreign body be in the eye.

In (4) a foreign body has almost surely entered the eye, and one must get a localizing X-Ray plate, to see whether the body be within the ball of the eye, or whether it has passed through into the orbit behind the eye ball, in which case it is usually harmless. If it be found within the eye it must be considered as a great source of danger, and it must be removed, if possible. If it be made of steel or iron, it can be removed by the electric magnet. If stone, glass, copper or brass, it is impossible to get it out, except in the rare instances, where the body can be seen with the ophthalmoscope, and grasped by a forceps thrust through a scleral wound. Particles attracted by the magnet can be removed through the Cornea, through the original wound or an incision made large enough to allow its escape, or through a wound in the sclera just below the scleral attachment of the external rectus muscle.

In the treatment of serious injuries of the eye, one must be constantly on the look out for the sort of a wound which makes the injured eye a menace to the good eye. Any penetrating wound, operation, or injury, can cause sympathetic inflammation, but most cases come from wounds which are not healing kindly, because of infection, or some impediment to healing, such as iris, lens matter or capsul, ciliary body, or foreign body in the wound. It is always wisdom to protect oneself by a consultation, in the case of a penetrating wound, in which after 10 days or two weeks the eye is not doing will. By this is meant, the eye is sensitive to light and to touch, it waters freely, the eye ball is soft and evidence of inflammation (ciliary injection) are not showing signs of abating. The patient should be warned of the danger of sympathizing inflammation, and given the opportunity of sacrificing the menacing eye by excision. A few will not run any risk whatever, in which case the doctor can free his patient and himself of a lot of anxiety, by excising the eye. More, however will choose to keep a dangerous eye in hope of it getting well, in which case the doctor has an anxious time ahead of him. He is wise to not take too much responsibility in acceding to the patients wish, and in the case of a minor, at any rate to get a written statement from the parents or responsible persons, in which these persons accept the responsibility of keeping a dangerous eye, against medical advice.

So long as an injured eye shows signs of being irritable without being tender to touch, easily made red by exposure, or use, it must be considered as a possible menace to the fellow eye.

An eye which contains a foreign body, may produce sympathetic inflammation months or even years after the injury. It is thus wise to warn the patient to keep in touch with medical advice for a considerable time after a serious injury, so that he may receive assistance in time to be of some service to him. He should be told to report promptly if any symptoms arise, as photophobia lachrymation, redness, a blurring of vision in the uninjured eye, as these are the earliest signs and symptoms of sympathetic inflammation.

Acidosis and Alkalosis.*

By T. BEATH, 3T0.

The following is a brief presentation of some of the concepts and empirical knowledge necessary to understand and treat acidosis and alkalosis.

In pure water the concentration of hydrogen ions is equal, and the solution is therefore said to be neutral, at an approximate normality value for each of $Nx10^{-7}$, i.e., the water is a solution of .ooooooo1N concentration of hydrogen and of hydroxyl ions, or in other words one gram molecule of water in each ten million litres is dissociated into one gram of hydrogen ions and 17 grams of hydroxyl ions. In any aqueous solution the greater the hydrogen ion concentration, the less the hydroxyl ion concentration and vice-versa. Excess of hydrogen ions over hydroxyl ions is present in a solution on the acid side of neutrality while excess of hydroxyl ions over hydrogen ions is present in an alkaline solution. Since the hydrogen ion and hydroxyl ions concentrations are opposite yet interdependent entities, it is sufficient and customary to speak of hydrogen ion concentration only in expressing acidity—its increased concentration meaning greater acidity and its decreased concentration meaning lesser acidity. The symbol "pH" is used to express the hydrogen ion concentration in grams per litre and its numerical value is the common logarithm of the reciprocal of this concentration. Therefore, the greater its value, the less the hydrogen ion concentration. Neutral aqueous solutions have approximately pH 7.0 at 25° C. [1].

Neutrality of the Blood.

The blood is an approximately neutral fluid (pH 7.3-7.5) maintained by two mechanisms, viz. (1) Buffer substances, (2) The execretory mechanism reality the blood is very complex, it may be regarded as a solution containing a certain amount of acid with a balancing amount of base, nearly all of which is sodium and potassium. The balance of acids and bases is frequently diagrammatically represented (Fig. I. and Fig. II.).

Chlorides.

The chlorine radical present in the body is in the form of chlorides which are neutral salts of the strong acid, hydro-chloric acid. Chlorides found within cells are mostly potassium chloride, those found in the body fluids are mostly sodium chloride. (In general, the sodium radical is found mostly in the extracellular fluids, while the potassium radical is found mostly intracellularly.) [12]. The union between hydrochloric acid and base forming chlorides is too strong to be broken by any acids which form biologically. This statement must be qualified however in noting that free hydrochloric acid derived from the body chlorides is secreted into

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*I am very grateful to Prof. H. Wasteneys, Prof. V. J. Harding, Dr. T. G. H. Drake and Dr. W. R. Campbell for very valuable advice given to me in preparing this paper.

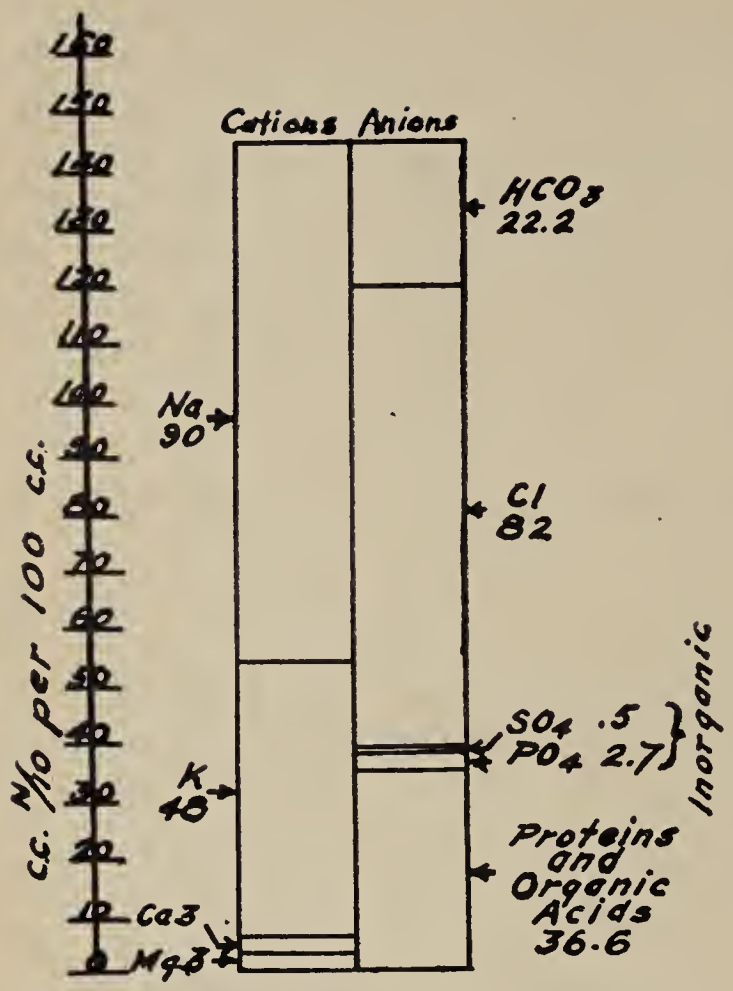

Fig. I. Acid-Base Balance in Whole Blood.

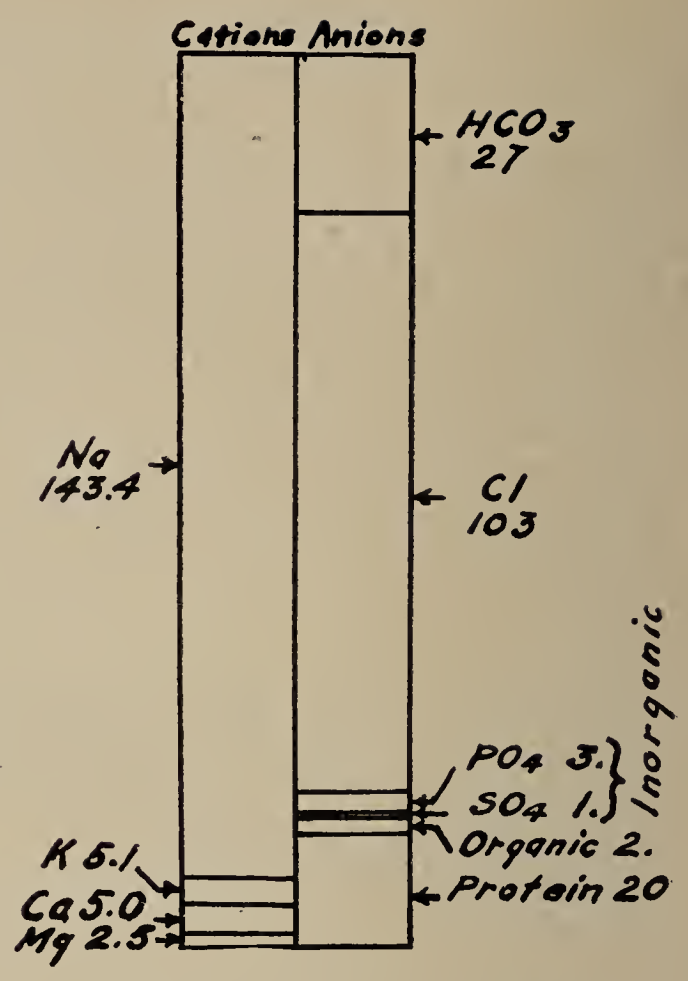

Fig. II. Acid-Base Balance in Plasma.[5]

FIG. I. THE ACID-BASE BALANCE IN WHOLE BLOOD.

Na, K, Ca and Mg. calculated from Kramer and Tisdall[3] (cf. Carl Schmidt) HCO_3 calculated by use of a nomogram[4] from the value in plasma of 27 c.c. sol. per 100 c.c[5] assuming volume of cells=50%, pH 7.38[6] and oxygenation=50%. Cl calculated from analysis of Norgaard and Gram[7 8]. Inorganic sulphates from Dennis[9]. Inorganic phosphates from an average between phosphates in plasma and corpuscles from analysis by Bloor,[10] assuming the valency of phosphoric acid as 1.8 (Bohr).[11] Proteins and organic acids are taken as the difference.

the stomach. Also noting that in the chemical fluctuations of the blood chlorine passes through the cell membrane of the corpuscles in both directions and in this passage it is not in the form of its sodium or potassium salts, as the cell membrane is relatively impermeable to both the sodium and potassium ions. [12]. In the blood 1-3 of the chlorides are intracellular.

Weak Acids.

It is an elementary fact in physical chemistry that in dealing with weak acids in solution the hydrogen ion concentration of the fluid is dependent upon the ratio of free acid to the salt of the acid It is an observed fact that the hydrogen ion concentration of the body fluids remains constant within narrow limits. The effect on hydrogen ion concentration of increased acids in the body fluids resulting from normal or abnormal metabolism, can only be combated by increase in base content or by decrease in acid content of the body fluids. Through its excretory mechanisms, viz., the lungs and kidneys, the body possesses an excellent apparatus for eliminating acids and thus keeping the ratio of acid to salt constant and coincidently maintaining the constant concentration of hydrogen ions.

Carbonic Acid in the Blood.

Carbonic acid is always present in the blood stream as it is a function of the blood to transport it from the tissues to the lungs. It is a weak acid and at the pH of the blood, viz., 7.3-7.5, about 19-20 of it is in the form of the

salt ($NaHCO_3$ in plasma, $KHCO_3$ in corpuscles) and about 1-20 of it in the form of carbonic acid H_2CO_3 in simple solution. As a weak acid, the proportion of free carbonic acid to the salt is a function of the pH, i.e., they are mutually dependent. Roughly one third of the carbonates and carbonic acid of the blood is found in the corpuscles and two thirds are found in the plasma. The quantity of carbonic acid (free and as salt) carried in the blood stream is dependent upon and directly proportional to the base, Na and K, available to form bicarbonate. The total quantity of carbonic acid in the plasma is a good measure of the quantity in the whole blood and is called the carbon dioxide combining power of the plasma. It tends to remain constant (extreme normal values of 53-78 vols. per cent.) [13]. but its decrease means a decrease in the efficiency of the blood to transport carbon dioxide from the tissues to the lungs.

The Proteins.

The proteins also are weak acids and accordingly at any given hydrogen ion concentration, a definite proportion is combined with base. At the pH of the blood, the proteins as a whole are about half in the form of the salt, B-proteinate and half in the form of the acid H-proteinate. In this system composed of weak acids and the salts of the corresponding acids, partly by virtue of the proximity of the ratio of $\frac{\text{H-Proteinate}}{\text{B-Proteinate}}$ to a $\frac{+}{+}$ ratio, rather large changes in the proportion may take place with only very slight coincident change in the pH of the solution. Practically this means that in the blood proteins may liberate a considerable quantity of base to neutralize normal or abnormal increases in acids with only very slight change in pH and also that they may combine with normal or abnormal increases of base with like trivial fluctuation in the true acidity. This maintenance of an approximately constant hydrogen ion concentration in spite of additions of acid or base, is termed "buffering" and in the blood, over 70% of the chemical "buffer value" is due to the proteins present. [14]. The amount of base which is contained in such systems at a given pH is a function of the strength of the acid. The protein haemoglobin possesses the valuable property that its strength (dissociation) is reduced in changing from oxy-haemoglobin to reduced haemoglobin. Thus, when the tissue fluids as a result of oxidation are most heavily charged with CO_2, which under other circumstances would result in an increase in hydrogen ion concentration, the oxy-haemoglobin has become reduced haemoglobin and thus is reduced in strength so that at the physiological pH range base is set free for combination with the carbonic acid. This preserves the ratio of free acid to salt of this acid which gives approximate neutrality. This mechanism is the most important buffer of the body. [14], [15]

Salts of Phosphoric Acid and Organic Acids.

The phosphates and salts of organic acids act similarly to the proteins but are relatively insignificant in their amount and action.

Excretion of Carbonic Acid.

When acids enter the blood stream they are buffered by base liberated from proteins. As this source of supply becomes exhausted the additional factor in the maintenance of neutrality, viz., the combination of additional acids with the base which is bound as bicarbonate, becomes increasingly effectual. (Base available for immediate neutralization of added acids constitutes the alkali reserve). [16]. This mechanism increases the ratio of free carbonic acid to the salts of carbonic acid in the blood which would in turn increase the hydrogen ion concentration but for the mechanism provided by the blowing off of excess CO_2 into the alveolar space of the lungs The mechanism is itself

excited by the presence of excess CO_2 in the blood. This process is a much more rapidly effective buffering mechanism than the excretion of acid salts by the kidney and is of peculiar value because it involves no loss of base from the blood. When acids combine with the base of the bicarbonate a decrease in CO_2 combining power results with the consequent tendency to carbonic acid accumulation in the tissues, including the respiratory centre. This minute increase in the CO_2 tension in the fluid bathing the respiratory centre is the physiological stimulant to greater ventilation of the lungs (deep, rapid respirations) and thus a blowing off of carbon dioxide. Physiologically this stimulation acts in exercise where the carbon dioxide production is increased, pathologically it acts in a state of low CO_2 combining power.

Excretion of Non-Volatile Acids.

Other acids than carbonic acid are ultimately excreted in the urine. In their excretion they carry also a considerable amount of base — which amount is greater when there is a large alkali reserve in the blood or smaller when the alkali reserve is low. For example, in a state of decreased alkali reserve, phosphoric acid will be more largely excreted as NaH_2PO_4 than as Na_2HPO_4. The kidney may also excrete the acid in combination with the ammonium radical NH_3—which is thought to be derived in the kidney for this purpose from urea. The ammonium radical does not neutralize acids in the blood stream. The kidneys are the excretory path of aceto-acetic and B-oxybutyric acids (ketone bodies) when they are formed in the body.

Definition of Acidosis.

Acidosis is a clinical term. It does not mean increased acidity in the sense that the reaction to litmus may be red, but is defined as a decrease in the alkali reserve to such a degree that the carbon dioxide combining power is below normal, this secondarily involving an alteration of respiration which may or may not be observable by ordinary clinical methods. Normal CO_2 combining power of the plasma is 53-78 volumes per cent; i.e., 53-78 c.c. of CO_2 gas at N.T.P. are liberated by strong acid from 100 c.c. plasma in which the CO_2 tension is of physiological value. (10% lower in young children) [22].

Dehydration.

Dehydration is a very important complication of acidosis or alkalosis. In part at least, the dullness, stupor, inelastic skin and dry tongue are due to dehydration. It is considered to be the chief cause of death in the serious cases of diarrhoea in infants in whom the acidosis itself was properly treated.

Dehydration is much more than a problem of loss of water. To understand dehydration is to understand the pressure effects, the permeability of the functioning of the kidney, the blood capillaries which are bathed by the intercellular fluids and the relation of proteins and salts to this permeability, the physical chemistry of the body cells and the distribution of the body ions, all these in relation to the distribution of water in the body fluids both extracellular and intracellular.

It can be stated that the osmotic pressure in tissues (tendency to take up water) is largely dependent upon the concentration in them of electrolytes (salts) and proteins. The difference in the osmotic pressure between the various fluids, which are separated by cell membranes, is largely dependent upon the presence of indiffusible proteins which act by virtue of their own osmotic pressure and by virtue of the peculiar distribution of electrolytes which they cause in accordance with Donnan's equilibrium. [12].

Of practical importance is the fact that loss of water and loss of electrolyte from the body are mutually dependent and coincident. Not only a loss of electrolytes but a loss of the normal balance of the various ions,

especially Na,K, Ca and Mg ions is believed to be of importance in disturbing the water balance. The loss of electrolytes occurs in various ways, (1) in the acidosis of diabetes by excretion with the ketone bodies, (2) in diarrhea by loss through the bowel, (3) in vomiting by loss either of gastric secretion alone or hepatic, pancreatic and gastric secretions. [17].

In treating dehydration, the electrolytes and their balance is to be restored. When NaCI solution is given electrolytes are restored and the kidney is relied upon to restore the balance by selective excretion. Administration of a solution such as Locke's is more effectual as Na, K and Ca ions are all supplied.

On the above basis, the presence of acidosis in certain conditions is explained.

I. In Diabetes Mellitus.

In Diabetes, in which the quantity of available insulin is small, the carbohydrate metabolism is decreased. To make up sufficient calories for maintenace fat metabolism is increased and the body stores of fat may be drawn upon. When the ratio of fat metabolized, to carbohydrate metabolized (in calories) becomes greater than five to one, the fat metabolism becomes abnormal (ketogenic) with the production of ketone bodies, viz., aceto-acetic acid, B-oxybutyric acid and acetone. These acids decrease the alkali reserve in two ways:

1. In their excretion they carry into the urine, in combination, a quantity of fixed base.
2. In being buffered in the blood stream the alkali reserve, i.e., the alkali for the $\frac{CO_2}{NAHCO_3}$ system, is used up.

This means a lowered CO_2 combining power with consequent disturbance in respiration and this is acidosis. A diabetic acidosis is quite mild until the CO_2 combining power falls below 35 vols. per cent.

II. In Cyclic Vomiting.

In cyclic (recurrent) vomiting in infants and young children, an acidosis is frequently produced according to a rationale somewhat similar to that for diabetes. The lack of carbohydrate intake leads to a low carbohydrate metabolism (the child's carbohydrate stores are small) and a high but faulty fat metabolism (the child's metabolic rate per pound is relatively high). Aceto-acetic and B-oxybutyric acids are poured into the blood stream more rapidly than HC1 is lost by vomiting and the result is the lowering of the alkali reserve, which is frequently severe enough to give a clinical acidosis. An acidosis is not produced in pyloric stenosis nor in other acute intestinal obstruction with vomiting in which no food is taken by mouth. Also these "cyclic vomiters" if simply starved during an abeyance of activity, more readily develops an acidosis than a normal child. It is therefore assumed that there is also an undefined factor which makes the metabolism in cyclic vomiting in infants particularly prone to develop abnormal quantities of aceto-acetic and B-oxybutyric acids.*

Severe vomiting of pregnancy seldom and probably never causes an acidosis even though aceto-acetic and B-oxybutyric acids, through carbohydrate starvation, are produced. The explanation is that the amount of acid vomited is about equal to the amount of abnormal acids produced.

III. Chronic Diffuse Glomerulo Nephritis.

In chronic nephritis an acidosis develops in certain cases. No exact single cause is found in these cases, but a combination of several factors operates. The demonstrable factors listed represents the failure of the kidney excretion in regard to acid radicals. [18], [?9].

1. Retention of chlorides is quantitatively important.
2. Retention of phosphoric acid radical plays a minor but significant rôle.

3. Accumulation of sulphuric acid plays a very minor rôle.
4. Organic acid accumulation — of some importance.
5. Failure of production of ammonium radical by the kidney for excretion of acid radicals—this results in the excretion of the bases of the blood and hence the reduction in the alkali reserve.

IV. Acute Watery Diarrhea of Infants.

Acidosis is present in the majority of severe cases of acute diarrhea in infants showing intoxication [22], [?9] i.e., fluid loss, (sunken eyes, depressed fontanelle and inelastic skin) with mental dullness, apathy and even stupor. Increased pulmonary ventilation in this condition is an extremely grave sign (over 70% die). No single cause has been determined but the following factors operate.

1. The great loss of basic radicals in the stools. Bases are secreted by the pancreas and liver and rapidly rushed through the intestines before absorption takes place.
2. The body dehydration is great, being concomitant with the loss of electrolytes; there is consequently too small an excretion of urine to eliminate the acids of the body.
3. Some observers consider that the intoxication which produces the diarrhea also causes a profound metabolic disturbance leading to the production of acids and even the disappearance of alkali reserve in excess of that accounted for by the production of acids.

Treatment of Acidosis.

Having understood the rationale of the production of acidosis the principles of treatment become nearly obvious.

I. Diabetes.

(a) Further production of ketone bodies is prevented and the ketone bodies already in the body are burned up by the administration of insulin, with carbohydrate enterally or parenterally if necessary.

(b) It was pointed out that, in the excretion of ketone bodies in the urine, guantities are fixed base also were excreted. It is permissible with great care, to give alkali to make up this loss. Sodium bicarbonate or sodium citrate may be given by mouth in dose of 10-15, or in coma, possibly up to 30 grams in 24 hrs., to an adult. In this treatment the CO_2 combining power must be followed as the urine may not turn alkaline to litmus until there is overdosage. Overdosage is liable to cause death. Citrate or bicarbonate must not be given subcutaneously.

(c) A large amount of water is to be supplied. This not only helps to combat the dehydration present but brings the kidneys into greater function, enabling them to readjust the great disturbance in balance of electrolytes and to increase the excretion of ketone bodies. This may be combined with administration of carbohydrates or of saline, or both parenterally.*

II. Cyclic Vomiting in Children.

Here also there is acid production (ketone bodies) which is greater than acid (HCl) lost in the vomitus.

(a) Carbohydrate (5% glucose) is given parenterally with or without insulin. This quickly burns up the abnormal acids. When this has been done, further giving of carbohydrate does not get rid of any more acids but prevents their further formation.

(b) Administration of NaCl parenterally in normal saline or Locke's physiological solution. When the abnormal acids have been excreted or destroyed, because of the loss of HCl in the vomitus, there may be left too much base in the blood for the acid Cl radical (see alkalosis below). Administration of NaCl replaces the lost

*It is somewhat preferable in diabetics not to give glucose solutions subcutaneously because of the danger of necrosis. Intravenously it is given not faster than 10 c.c. per minute to an adult.

Cl while any excess Na is excreted by the kidneys.

(c) Large quantities of fluid in the form of 5% glucose solution given parenterally, enable the kidneys to adjust the electrolytes to the body requirements and excrete ketone bodies. Best results are obtained by alternation of 5% glucose with saline.

(d) Later treatment — small frequent carbohydrate meals and removal of foci of infection to prevent recurrence.

III.—Nephritis.

Treatment of acidosis in this condition is not specific and is combined with the treatment for nephritis. Giving as much water as is compatible with kidney function is probably the best single procedure. Some comfort is given by administering enough base to relieve the patient's breathing and to turn the urine alkaline. In the development of acidosis because of an acute exacerbation of the nephritis ,the administration of base may carry the patient over this acute stage.

IV. In Diarrhoea in Children.

(a) In this condition the dehydration is the most important danger. It must be controlled. Tissue and blood dehydration depend largely upon a decrease of the electrolytes (salts) which has taken place and to combat dehydration these must be increased. Normal saline is useful, but it is better to use Locke's solution parenterally which supplies divalent Ca ions as well as monovalent Na ions and K ions. The kidney is relied upon to adjust the proportions of the electrolytes remaining in the body.

(b) The amount of fluid (5% glucose) administered in this condition is greater than in any other. It is necessary to make up for loss which has taken place and especially to supply enough for the elimination of acids and unnecessary electrolytes through the kidneys.

(c) Administration by mouth of a mixture of

1% Sodium Citrate....1 part,
Orange juice .....2 parts,
10% Glucose .....3 parts,

with the aim of supplying water, energy producting and ketone preventing substances and also base which is not balanced by Cl. The citrate is metabolized leaving Na to form bicarbonate and so help restore the alkali reserve.

Preoperative Administration of Carbohydrate.

This excellent practice helps to combat starvation ketosis and its consequent tendency to acidosis after the operation and prior to feeding and if given with considerable water, especially parenterally, it helps to combat dehyration. In critical cases this may mean success or failure.

Signs and Symptoms.

The most important clinical indication of acidosis is the change in breathing. In a mild acidosis, the change may not be noticed subjectively nor even objectively on a cursory examination though careful measurements of pulmonary ventilation may reveal its increase. In more severe cases (which are often complicated by dehydration), breathing is deep and somewhat more rapid, though the depth is the prominent feature. The breathing in pneumonia is rapid and shallow, in obstruction it is labored and stridulous. The chest rises and falls with each respiration. The breaths are heaving, often involving the use of the accessory muscles and are pauseless. The peculiar "air hunger"—a breath which looks like a modified yawn, may be observed. The most striking feature is the amplitude of the respirations and the distinct effort with which they are accomplished. There is no cyanosis but often pallor, though on the other hand there may be a cherry red color to the lips. Drowsiness is often a prominent symptom and may become stupor. This is particularly so in cases in which de-

hydration is severe or where there is excessive production of acetone bodies. Only when there is accumulation of acetone bodies in the blood will the peculiar sweet odor of acetone bodies be detectable on the breath. While cyclic vomiting in children and acidosis are often concomitant, the vomiting is not caused by the acidosis nor, as was pointed out, is it the sole cause nor probably even the major factor in the production of acidosis. (21). In cyclic vomiting the first symptoms may be dated to only a few hours before a severe acidosis is found.

Diagnosis.

Acidosis of any severity is seldom found clinically except as a complication in diabetes, cyclic vomiting or acute diarrhoea in infants and chronic nephritides. The history or diagnosis of these conditions is usually easily obtained and acidosis may be predicted as a complication. A presumptive diagnosis is made when in any of these conditions there develops hyperpnoea as described. The presumption is verified when the CO_2 combining power of the plasma is found to be below 53 vols. (10% lower in young children).

Alkalosis.

Alkalosis is a condition exactly the reverse of acidosis. It is produced by a decrease in total non volatile acid radicals without a corresponding decrease in fixed base e.g. Cl in vomiting with loss of HCl or by increase in basic radicals e.g. in the over administration of base in the treatment of a case of acidosis or in treatment of a case of ulcer by the Sippy method. When Cl is lost from the blood stream in vomiting, base, with which it was combined, has to be neutralized. This is done by retention of the volatile acid, viz. carbon dioxide and the result is the formation of more bicarbonate in the blood which of course means greater carbon dioxide combining power. The upper limits of normal CO_2 combining power of the blood plasma is about 78 vols. per cent. (10% lower in infants and young children).

1. Alkalosis in Pyloric Stenosis.

Alkalosis is liable to be produced in an obstruction in which there is great loss of HCl by vomiting the gastric juice (e.g. congenital hypertrophic pyloric stenosis). In pyloric stenosis, the more complete the obstruction and the greater its duration, the greater the degree of alkalosis. With the obstruction at the pylorus ,the alkaline intestinal contents are not lost to the body. The production of alkalosis depends upon there being a greater loss of HCl by vomiting than there is production of acetone bodies, because of carbohydrate starvation. Ketogenesis is not so great as in cyclic vomiting (because of the inherent tendency for ketogenesis in cyclic vomiters and because some carbohydrate does go past the pylorus in pyloric stenosis) but is nearly enough in some cases to replace the HCl lost in vomiting. In pyloric stenosis then, there is a loss of Cl from the blood leaving some excess Na. This excess Na may be balanced in part by aceto-acetic and B-oxybutyric acid formation. That which is not balanced by these non volatile acids increases the CO_2 combining power by forming bicarbonate. If, in this condition of border line alkalosis with ketosis, a small amount of 10% glucose were given subcutaneously, the acetone bodies would rapidly disappear, with possibly a precipitation of a condition of alkalosis.

Treatment.

Rational treatment is the administration parenterally of quite large quantities of normal (.9%) saline (to supply Cl, relying on the kidneys to excrete Na), alternated with 5% glucose to supply fluid for the use of the kidneys and keep down ketosis (acetone bodies being in themselves toxic).

Alkalosis in Treatment.

(a) Alkalosis may also be produced by too rapid reduction of the ketosis in cyclic vomiting.

The treatment is the same as for the alkalosis in pyloric stenosis.

(b) It may be produced by giving too much alkali to any patient e.g. in treating acidosis in diabetes. Overdose of alkali is liable to cause death.

The treatment is to give large amounts of fluids. In adults ammonium chloride may be given per rectum but this is not done in infants as retention in the bowel is poor.

Alkalosis is not a common condition but may be a very serious one. The clinical feature is the decreased pulmonary ventilation shown by quiet, slow and shallow respirations. The respirations must be observed when the patient is quite free from excitement as in infants especially, slight disturbance will produce respirations in alkalosis which are not abnormal. It is to be noted that there may be alkalosis with ketosis and that ketone bodies in the urine do not justify treatment by giving sodium bicarbonate, etc., as for acidosis.

Provisional diagnosis may be made in possible cases with a decrease in pulmonary ventilation. The diagnosis is certified by the estimation of the CO_2 combining power. Vomiting of pregnancy never produces acidosis and rarely produces alkalosis.

There are certain cases in which the diagnosis between acidosis and alkalosis is in doubt, and therefore the treatment is in doubt. For example, in complete obstruction at the pylorus here may be marked alkalosis with ketosis; or too active specific treatment of a case of acidosis may lead to an alkalosis. Since alkalosis is a dangerous condition and since its treatment is largely non specific, it is safer to treat a doubtful case non specifically as a case of alkalosis i.e. parenternally give Locke's solution or .9% Na C1 solution alternated with 5% glucose solution and rely upon the selective excretory power of the kidneys to properly adjust the radicals reained in the body.

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Decerebrate Rigidity And Its Clinical Aspects.

By W. A. HAWK, 3T0.

(Introduction)

In order to attempt a discussion of this subject we will divide it into two parts—(a) Decerebrate rigidity in animals or experimental rigidity and (b) Decerebrate rigidity in man or clinical rigidity.

On the former aspect numerous well written papers have occured in the last decade—from these generalizations and deductions have been made and as a whole the experimental conditions now stand on a firm and well established basis.

On the contrary little attention has been paid to the clinical side and we have but isolated case reports to draw upon. From this it will be seen that the latter part of the paper will be rather weak, its intention however is not so much to present a definite system of laws or deductions but to stimulate discussion and thought on this subject.

Experimental Aspects of Decerebrate Rigidity.

(History)

As is customary we will begin by a brief history of experimental decerebrate rigidity:—

Sherrington noticed (1896) that the removal of both cerebral hemispheres caused general rigidity of the body. Since then many authors have contributed to the explanation of the phenomenon.

Sherrington called this rigidity "Decerebrate" and pointed out that it was not caused by the action of pyramidal tracts (excitation or inhibition) for the cutting of the pyramidal tract of one side at the level of the medulla did not influence the rigidity of the opposite side. Nor was the rigidity influenced when the posterior tracts were cut at the same level.

An increase of rigidity appeared when the medulla was irritated by a faradic current. These experiments were continued by Horsley and Thiele. They found that the removal of both hemispheres leaving the thalamus intact did not cause rigidity.

This animal (thalamic animal) has a normal muscle tonus and can stand by itself and change its position under the influence of external stimuli.

Radimaker has determined the different parts of the nervous system which governs the muscle tonus. He confirmed the experiments of Horsley and Thiele—additionally showing that a portion of the thalamus is sufficient to keep the muscles in a normal tonus. Rigidity however appears when the mid brain is cut at the level of the red nucleus. From this he drew the conclusion that the red nucleus is the mesencephalic centre of normal muscle.

It however remained to be shown where the centre of decerebrate rigidity is located. Wilson thought that any cause which destroyed the influence of the cortex in the mid brain and in the cerebellum makes the latter develop symptoms of decerebrate rigidity. Sherrington however proved that decerebrate rigidity may continue after cerebellar excision. Brown then showed that irritation of the mid brain causes decerebrate rigidity even in cases where the cerebellum is absent. In cases studied by Weed where the cerebellum was removed previous to decerebration, rigidity appeared, but only for a short time. The author concluded that the center of rigidity lies within the mid brain probably within the red nucleus.

Magnus found also that decerebrate rigidity may appear in animals deprived of their cerebellum, and that such when produced does not disappear with the removal of the cerebellum.

This then apparently concludes that the center of rigidity does not lie within the cerebellum. A symposium of authors showed that decerebrate rigidity appears not only when the mesencephalon is cut at the level of the red nucleus; but also when the cut is made lower down across the medulla oblongata as far ts the nucleus of the VIII. nerve. A cut below this level causes flaccidity. Horsley, Thiele, Bazett and Penfield thought this center lay within Deiters nucleus.

John Hunter in 1924—tried to verify the supposition experimentally—he destroyed in the decerebrate and rigid animal Deiters nucleus without any change in tonus. A cut across the medulla at the line of union with the pons caused flaccidity of the muscles.

From the above experiments we see that the center lies somewhere within the mid brain between the level of the red nucleus and the lower part of the medulla.

The most recent article at present on this is probably that of Zandt and we see that the inferior olivary nucleus is responsible for decerebrate rigidity. This is confirmed by the fact that decerbrate rigidity disappears when the olives are destroyed—and also by the fact that in clinical cases of decerebrate rigidity the olives are well preserved but their communication with the higher centers destroyed.

(Experimental Picture)

The first thing before we analyze the nature, etc., is to give a brief picture of an experimental case of decerebrate rigidity. In the following description we have a decerebrate cat—section was made through the caudal portion of the mid-brain. In these circumstances all the muscles which maintain the animal in a standing position are in strong tonic contraction—these include the limb extensors—elevators of the head, neck and tail and extensors of the spine—utilizing Sherrington's so-called "antigravity" muscles. The rigidity is an exaggeration of normal tone and under the law of reciprocal enervation we accordingly find the antagonistic flexor groups of muscles are inhibited and atonic. In posture the head is upheld—spine fully extended—tail maintained horizontally and the limbs fully extended. The upper limbs are retracted at the shoulder—forefeet held with the palmar surfaces directed ventrally as in standing. The hind legs are similarly held. Such an animal if carefully placed in its feet can stand. It breathes automatically with a normal rhythm and showspseudo affective and other reflex reactions. As the section is caudal to the thalamus we have a loss of heat regulation and it lives but a few hours if it is not kept in an incubator. If, under these conditions it be kept alive for several weeks, the rigidity is maintained throughout, but may be replaced from time to time with a plastic flexor rigidity.

Such an animal though it can passively stand is unable to walk, run, or jump, and in addition is quite unable to right itself if placed in an abnormal position.

(Postural Reflexes)

In attempting to understand the basal factors of physiological value underlying the condition we must say a few words on postural tonus and the reflexes maintaining it. All the higher animals normally exhibit in repose or action characterictic attitudes through which they are orientated in regard to space. When such is displaced it tends to return and to any such displacement attempted there is a resistance by the muscles of the animal.

Such static postures are maintained for long periods of time without fatigue. There is a state of tonic contraction of the muscles concerned, modified and produced through the channels of the central nervous system.

Postural reaction occures at rest and in motion—the former are static the latter stato-kinetic. The reflexes which maintain the body in a fixed position are those of pose, those restoring

pose after displacement are righting reflexes.

According to the work of Magnus and de Kleijne at Utrecht the maintenance involves chiefly reflexes from the neck muscles, the eyes and the labryinth. Similarly the righting reflexes are maintained through these centres.

We might give a few practical examples of these facts—if the neck be moved so the head points to the left, we have in the left side an increased tonus and a reciprocally inhibited tonus on the right. Similarly if the head be bent back, the cat assumes a position of looking up at a shelf—extended forelegs, flexed hind legs.

Rotation of the animal brings in the labyrinthian reflexes—for instance the tonus is greatest when the animal lies on its back—least when lying on its belly. These are of value in the righting reflexes, tending to keep the head in its normal position no matter what the position of the body is.

In the statokinetic reflexes changes in the rate of movement—angular acceleration—rotation, etc., produce reaction through the labryinth. These reflexes tend to bring the body into normal pose which then is maintained by static reflexes.

To sum up briefly, the control of postural tonus is through

(a) Local spinal arc of tonus,
(b) This is controlled by the subcortical centres of unknown location—probably in mid brain or medulla.
(c) These are influenced by reflexes from the neck musculature, labryinth and eys.
(d) A general control of these centres is exerted through a thalamic centre.
(e) Voluntary control of all occur through the cortical centres.

With these facts in view, let us amplify them and attempt to find the physiological basis of decerebrate rigidity.

(Etiological Principles).

In attempting to understand the nature of decerebrate rigidity we believe now that it is essentially a reflex manifestation, these are proprioceptive afferent stimuli which arise in the muscles through specialized nerve endings —the rest of the reflex arc is through propriospinal neurones to the anterior horn cells. Through this we obtain a general postural background which has been shown capable of definite modification by afferent stimuli from various portions of the body—labryinth, etc.

(1) According to Liddell, if any of the antigravity muscles are stretched we have ensuing a reflex contraction opposing the stretching force—these so-called "stretch-reflexes" are limited to that portion of the muscle under tension. These reflexes oppose gravity and are probably the essential basis of decerebrate rigidity.

(2) According to another author it is probably due to two causes—(a) interruption of the corticospinal tract at some part of its course, and (b) concomitant interference with the brain stem mechanisms concerned with the maintenance and regulation of postural tone or of the tracts in relation to them.

(3) According to Walshe—which is apparently the most tenable of theories regarding the nature and cause of the rigidity we postulate.

(a) The reflex arc of muscle tonus.

(b) A series of brain stem centers and paths extending from the anterior portion of the midbrain to the medulla —and whose purpose is to adjust the muscle tone to the various motor activities of life It may be reasonable to assume that the rigidity is the result of the unfettered action of the local spinal reflex arc of muscle tonus. In addition to removal of cortical control we have abolished subsortical control.

(Centers Involved)

Another great controversial point amongst physiologists is the location of the nerve centers involved. Our present day knowledge of the nerve centers involved is

1—Cerebellum in definitely not involved

2—Essential centers are now (1928) believed to be

(a) Between entry of VIII nerve and upper cervical cord, center for labyrinth and neck reflexes except righting reflexes.
(b) Above this level to the nuclei for the eye muscles lies the labyrinthian center for control of the eyes.
(c) Still higher in midbrain is the center for the righting reflex.

3—The center for decerebrate rigidity was believed to be in the medulla near the calamus scriptorius.

4—Graham Brown believed that the red nucleus was concerned with the normal tonus distribution and the righting reflexes, that it tends to counteract the rigidity by throwing the flexors into action. Opposed to this was our medullary center.

5—The most recent work as previously quoted believes that the inferior olivary nucleus is the medulla is the main factor.

The qualifications for decerebrate rigidity to summarize briefly are—

(a) Intact reflex arc of muscle tonus.
(b) Interruption of the cortical and subcortical influence on the local arc. This may be done by transverse section through the brain stem between the mid brain and medulla. Clinically we may see it in degenerative lesions effecting the brain stem in these areas—more details will appear later.

Clinical Aspects of Decerebrate Rigidity.— —

(History)

Going back into history we find that probably the first expression of the possibility of decerebrate rigidity in man was brought forth in 1901 by Collier and Buzzard. In 1905 Thiele considered spasticity in man as a corresponding condition but brought forth noi clincal evidence to support his views. A few years later in his Hughlings Jackson lecture—Victor Horsley correlated spasticity in man with the work of Sherrington.

The marked rigidity of decerebrate conditions was reported also by Antheaume in 1920—a case of general paralysis in a patient with catatonia, catalepsy and stereopsy. In 1923 Urechia and Eliher reported 4 cases, and other authors have reported on the relationship of decerebrate rigidity to general paralysis.

(Clinical Picture)

The picture of decerebrate rigidity in man is somewhat similar to that in animals but shows some material differences—as a consequence we shall detail a typical clinical picture—the extremities showed a flexion contraction of both arms, hands and fingers—marked adduction of both upper arms with definite pronation—fingers tightly flexed—legs held rigidily in extreme extension with feet plantar flexed and the legs were forcibly adducted. The arms and legs could be placed in various positions which would be well maintained for some time gradually coming back to their original state (n.b.) the flexor contraction of the upper extremity is not a picture of true extensor rigidity.

Considerable work has been done by various writers to explain the flexion position of the upper arms—the most recent, that of Brain, will be given as the most logical:—

Brain has recently shown that such a flexion will assume an extension posture of the patient will assume a quadrupedal position (i.e. that normally assumed by any four-legged animal). Such show the Magnus and de Kleijne reflexes and consequently he draws the conclusion that this is a definite decerebrate rigidity—the difference in posture assumed from a decerebrate quadruped is linked with the various evolutionary changes in assuming the bipedal position—so in a decerebrate ape we have an almost similar picture—the

upper limb assumes a position of prehension, the lower one of locomotion.

(Diagnostic Features)

In order to guide the student or practitioner in his clinical studies we must give further and more limiting diagnostic points about decerebrate rigidity. The chief characteristics are:-

1—Quality of the rigidity—it is a true hypertonus of the kind familiar to us in spastic hemiplegia. It may be abolished by posterior root section. It may present the quality of plasticity—as seen by the tonic prolongation of the tendon jerks and the shortening and lengthening reaction—which is seen in the sudden yielding of the rigidity on passive stretching of the muscle, and the adoption of a new and increased length — clinically the clasp knife rigidity.

2—We have given a picture of the typical example seen—most commonly the flexion of the upper extremities.

3—In view of recent work we should now obtain the Magnus and de Kleijne reaction, i.e., upon moving the head to one side we find increased tonus and extension in the muscles of the extremities in that side, and diminished tonus in the opposite side.

Probably the main condition with which it may be confused is seen in the tonic fits of tetanus and strychnine poisoning—in these cases due to an overthrow of the reciprocal inhibition plus a hyperexcitability of the neurones. Consequently we have a generalized spasm of the sketetal muscles in contradistinction to the selective spasm with reciprocal inhibition seen in decerebrate preparations.

(Occurence)

We shall now take up a brief study of the conditions in which decerebrate rigidity has occured—we shall begin with—

A—Trauma.

We have a case report by Wayne Babcock of a football player who dove into a scrimmage and was found immediately afterwards unconscious. He lay on his back—arms in rigid contraction—legs rigid — rarely showing a transient clonic phase. Coincident medullary damage produced changes in pulse and respiration.

This case was described by the authors as decerebrate rigidity—it differs slightly from the true clinical picture.

B—Encephalitis-Lethargica.

Occasionally the rigidity that occurs after epidemic encephalitis is of such marked degree as to make it the outstanding feature simulating decerebrate rigidity. From the article by Weisenburg Alper:—

We go now to a brief resumé of several reported cases of marked rigidity in encephalitis. Hunt describes a case—the association of general muscular rigidity of the paralysis agitans type with the peculiar lethargy characteristic of epidemic encephalitis produces a clinical picture closely resembling catatonia. Bassoe states in severe cases of encephalitis the whole body becomes rigid—movements slow and balanced — the condition resembling both catatonia and advanced Parkinsonian rigidity. Petrin and Brahme showed two cases of total immoblizations of the extremities through hypertonia following encephalitis. In the first case the rigidity was so great that passive movements were impossible—only respiration, swallowing and eye movements were possible. Musson and Bennett report a similar case in which autopsy showed disease of the thalamus and slight involvement of the corpus striatum—the substantia nigra was markedly diseased—this is one of the few cases with a pathological report localizing the lesion.

In certain cases of encephalitis they were able to locate lesions in the nucleus ruber when which were then considered responsible for the extreme rigidity.

C—Epilepsy.

Probably most of the recent work was done on the relationship of decerebrate rigidity to epilepsy, many cases in the tonic stage assume a picture quite similar.

Case of chronic hydrocephalus with epilepsy. The patient fell with legs and arms slightly convulsed which soon stopped—he lay on the ground legs stretched and close to each other—arms by his side and extended. With some force the extension of the limbs could be overcome but in it being withdrawn there was a resumption of their former position. At autopsy there was shown a marked internal hydrocephalus.

A text book description of the tonic stage states—the muscles are rigid—head is retracted, upper extremities are either extended or flexed, hands clenched—trunk rigid—lower extremity extended but may be partially flexed. The spasm lasts only a few seconds rarely ever 30 seconds.

We shall give a brief abstract of the most recent article—that of Lennox and Cobb:—

In the etiology of epilepsy we have four theories at present—amongst these is,

The "Release" theory suggested by our recent studies on decerebrate rigidity and locomotor reflexes. This theory holds that the convulsions come from a temporary suspension of function of the higher centers which allows the lower centers to discharge explosively.

Martin states: "Thus on analysing the full epileptic fit I have not found any unequivocal evidence of cortical stimulation but on the contrary in the prodromal symptoms in the unconsciousness and in the presence of an extensor plantor response at the end of the clonic stage I see indications of cortical inhibition, the movements of the clonic stage may be explained either by cortical or by brain stem and spinal action but the latter explanation is much more in accordance with the other elements of the fit."

Martin claims the prodromal apathy as an indication of cortical inhibition. The majority of the cases in the tonic stage show a picture roughly resembling a decerebrate preparation. The clonic movements of the legs are due he claims to spinal reflexes like the walking of a decerebrate animal, there has been a passage from true decerebrate rigidity to a picture seen in the "thalamic" animal with its walking reflexes.

Yahovlev notes that in minor attacks the babinski may be elicited from the beginning of the attack, symptomatic of interference with the corticospinal tract.

D—Spastic Hemiplegia.

Walshe in particular considers the two to be identical. A typical picture of spasticity very closely corresponds to that seen in decerebrate rigidity, viz:—the lower limbs lie fully extended and plantar flexed—the extensors show increased tone plus brisk tendon reflexes. We see the clasp knife rigidity—prolonged tendon jerks. The flexor muscles show decreased tone. The spasticity might be modified by reciprocal inhibition—seen in the flexion and crossed extension reaction.

Such patients are of course not wholly decerebrate and man does not show the results of a complete transection of the cord.

Walshe believes that the lesion of the corticospinal tract produces secondary changes in the sub-cortical centers and thus produces the rigidity.

E—Amaurotic Family Idiocy.

The only material on this relationship is in a case report by Ham:—

Amaurotic family idiocy—girl apparently normal till about 3 years—since then various progressive degenerative conditions have occured. Amongst the pathological lesions noted at necropsy were—degeneration of the ganglion cells of the nucleus ruber and

atrophy of all cerebellar layers. The outstanding clinical feature was the decerebrate rigidity — this however differed from the picture on experimental animals in the attitude of flexion in the upper extremity. The theory of Walshe that true decerebrate rigidity entails an involvement of the pyramidal tract was not shown here—the causative agent probably being the damage to the nucleus ruber; disregarding on recent theories the atrophy of the cerebellum.

F—Progressive Lenticular Degeneration.

In such a case of hepatolenticular degeneration wherein a cirrhosis of the liver is accompanied by a progressive lenticular degeneration—here we have evidences of hypertonia simulating a decerebrate rigidity.

G—General Paralysis of the Insane.

Many authors believe that the catatonia seen in this state is related to decerebrate rigidity. In 1923 Urechia and Eliher reported 4 cases with generalized rigidity and lesions in the globus pallidum—more recently Stock reported the occurence of catatonia in 14 of 19 patients with general paralysis —lesions in the corpus striatum.

H—Dementia Praecox.

Many authors suggest such a relationship but a study of recent cases and articles does not show any explanations or theories. The catatonic state shows such a marked disimilarity to our clinical picture of rigidity that we must doubt the association.

(Conclusion)

1. Decerebrate rigidity is a symptom seen in various diseases of the central nervous system.

2. We are able to postulate the requirements for clinical diagnosis.

3. The lesion must be a relation to the sub-cortical centers involved in experimental preparations.

4. The exact pathogenesis of the lesion is quite variable.

5. We are able to divide clinical symptoms into 4 groups, on locations of the lesions in the brain.

(a) Flaccid paralysis—below medulla.
(b) Decerebrate rigidity — below thalamus.
(c) Thalamic animal — below cortex.
(d) Normal cortical control.

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Breech Deliveries.*

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According to statistics, breech presentation occurs in three percent of all labors. The foetal mortality in such cases is about three times greater than in cephalic presentations, namely ten to fifteen percent In the Sloane Hospital series, 1920 to 1922, the percentage of viable babies—born dead or dying in the first few days of life—was thirteen percent Spinal cord haemorrhage was noted in forty-seven per cent, and fractured vertebrae in thirty-six percent. Intercranial lesions were found in twenty-five percent of these babies, while asphyxia alone accounted for only five percent of these deaths. Abnormal pelvis was present in thirty-five percent of these cases and fifty percent of the mothers were primipara. The incidence of maternal morbidity was greater than in cephalic presentations due to the extra manipulation and trauma associated wit ha difficult delivery.

Breech presentations therefore take a place in obstetrics as one of the great causes of stillbirth, invalidism among women, and subsequent sterility. The clinical course of labor in which the breech presents, is slower than in cephalic cases, because the soft breech does not excite strong pains as the hard head would do. The membranes often rupture early, and dilation of the cervix is slow due to the absence of its natural dilator. Prolapse of the cord may occur, also compression of the cord, after the liquor amnii has escaped. Occasionally the placenta separates during the delivery of the head, sometimes a retraction ring forms in long drawn out labors.

Delivery is further complicated by the presence of a contracted pelvis, or a rigid pelvic floor. Add to these possibilities, undue haste on the part of the operator, perhaps attempting to deliver the head through a partially dilated cervix, and tragedy is bound to follow.

In order to cut down these percentages of foetal death and give a baby presenting by the breech a greater chance of being born alive, two courses are open:

First—external cephalic version in the later weeks of pregnancy, or

Secondly—perfecting oneself is the art of breech delivery.

The former method is highly recommended and practised by most obstetricians, and is a life-saving measure when any disproportion exists between the foetal head and maternal pelvis. Unfortunately, a breech presentation is not always easy to diagnose, especially in a primpara, and often we are unconscious of the presentation until the patient is in active labor, and membranes are ruptured. Diagnosis can usually be made by means of careful abdominal examination, in which a ballottable mass is found in the fundus. The foetal heart is usually on a level with, or above the umbilicus, but if the breech is well down in the pelvis, the heart sounds are heard below the level of the umbilicus, and a cephalic presentation may be suspected. On rectal examination, the presentating part is usually high in the pelvis and a frank breech may easily be mistaken for the head. However, if the examining finger is carefully swept from side to side, variations in the consistency of the mass can be distinguished, the sulcus between the two bony prominences can be identified, or the hard pointed tip of the sacrum recognized. An irregu-

*Reprint courtesy St. Michael's Hospital Medical Bulletin.

lar surface should put one on guard for a complete breech with the feet lying alongside the buttocks. If in doubt, a vaginal examination should allow a positive diagnosis to be made.

In supervising a case of breech presentation, a policy of watchful expectancy should be observed. The patient is kept in bed to preserve the bag of waters as long as possible, and delivery should not be attempted, or allowed, till the cervix is fully dilated. This last rule is often broken unconsciously by the patient herself who bears down before the second stage, and may deliver the breech of a small baby. The after coming head is then caught in the rim of cervix and the child is damaged or the cervix lacerated. If all conditions are favorable, the pelvis ample, perineum relaxed, contractions good and the baby not large, spontaneous delivery may occur, so every opportunity should be given nature to accomplish this end, aided only by gentle pressure from above, and a guiding hand below. But more often, complications ensue, and our problem tonight is to review the methods used by well known obstetricions in overcoming these difficulties.

Most writers are agreed that four courses are open when a frank breech reaches the pelvic floor and no progress is being made:

(1) The use of a fillet in the groin for the purpose of traction. This method is falling into disuse.

(2) Traction by means of finger placed in each groin, using no undue force.

(3) Forceps applied to the breech. This method is recommended by Lee and Munro Kerr with the warning that a good application is sometimes difficult and the forceps may slip.

(4) Bringing down a leg after "breaking up the breech" by the Pinard manoeuvre. This necessitates an anaesthetic and is accomplished by inserting a hand alongside the thigh, flexing the knee by pressure in popliteal space with the index finger, then slipping the hand along the leg to seize and deliver a foot. At times this seems like an impossible task, but De Lee claims that with perseverance it never fails.

Having released a foot, the breech will now descend, and if traction is used to deliver, it should be used coincident with the uterine contractions. When the umbilicus appears at the vaginal outlet, it may be necessary to hasten the delivery without sacrificing gentleness or skill. Traction is affected by grasping the child with the thumbs over the dorsal aspect of the pelvis, and the fingers over the anterior surface of the thighs. When the angle of the scapula appears, the child is carried forward, and towards the side to which its abdominal aspect is directed, a hand is inserted along its back to the shoulder, and the posterior arm is released by traction of the fingers in the cubital fossa. If the other arm cannot be quickly disengaged in its anterior position, the body is rotated and delivery posteriorly is repeated.

To deliver the head, the baby's body is allowed to rest astride the operator's arm, two fingers of the hand being inserted well back in the baby's mouth —not for purposes of traction, but to insure the maximum flexion of the head. Two fingers of the free hand are placed across the baby's shoulders and gentle traction downward and backward brings the nape of the neck underneath the pubic arch. Forward and upward traction allows the suboccipital area to pivot on the pubic bones and the baby's face appears at the vulva. Great control should be exercised here to prevent sudden expulsion of the head, causing intracranial lesions and also perineal damage to the mother. If forceps are used to deliver the after coming head, they must be near at hand so no valuable time will be lost.

When a frank breech is arrested at the inlet the same manoeuvres can be

carried out, but we are warned to release the anterior leg, because the anterior buttock will catch on the symphysis if the posterior leg is brought down first. As the breech approaches the perineum, forward traction on the thigh will allow the other buttock and leg to slide over the pelvic floor. As the child descends it is rotated so that the back is anterior and in the transverse diameter of the outlet.

Williams is in favor of delivering the arms when the scapula has appeared at the vulva. This is done by rotating the body, with digital pressure on the shoulder girdle, bringing one arm under the pubic arch and releasing it, then rotating the body anteriorly to disengage the other.

Eardly Holland, while advocating conservation in the handling of breech deliveries, advances an argument which is applicable to certain cases. He mentions that if progress of a high frank breech is delayed in spite of good uterine contractions, one is justified in bringing down a leg. Unless this is done there is danger of the breech becoming impacted in the pelvis, and close retraction of the uterus will make difficult the subsequent delivery, or cause asphyxia of the baby.

A discussion of breech deliveries would not be complete without reference to the work of Potter of Buffalo whose name is so closely linked with that of version and breech extraction. I will not discuss versions here tonight, except to say that it is a necessary part of the obstetrician's training, and confidence in your ability to do a version and breech extraction will often convert an impossible situation into a safe delivery. I refer particularly to abnormal presentations such as face, brow, shoulder, transverse, high posterior positions, unengaged heads due to a flat pelvis, and placcentae praevias.

Briefly, Potter's technique for doing a breech extraction differs from the orthodox teaching in his advocating the rotation of both shoulders anteriorly without inserting a hand in the vagina. When the breech is delivered, downward and backward traction is used till the whole scapula shows plainly. He then rotates the baby's body, holding it by the scapula and thorax. As one shoulder appears beneath the pubic arch, he places a finger on the shoulder, slides it down to the cubital fossa and disengages an arm The baby is then rotated until the other shoulder appears anteriorly, and the operation is repeated. He claims that even though the arm is extended, the act of rotation causing it to brush against the pelvic wall, will bring it to a right angle from the body.

No abdominal pressure is used by Potter till the arms are delivered, as he claims it may push the head between the arms and thereby result in their becoming extended. When the flexion of the head is controlled by inserting the finger in the mouth, suprapubic pressure is applied, directed downward towards the floor to help engagement of the head, then as traction is applied to the shoulders, pressure is used in the axis of the pelvis.

Several points in Potter's technique are worthy of note—it is the correction of all factors that makes the operation a success:

(1) Position of the patient is of the utmost importance. He never delivers one on the flat but always across the bed in a modified Walcher position, the legs held by two nurses or supported by a couple of chairs. This allows greater relaxation of the perineum and provides a longer true conjugate of the pelvis.

(2) The cervix must be fully dilated. The bladder must be emptied of all urine.

(3) In performing a version, deep anaesthesia with chloroform gives better relaxation. In extracting the arms and head after the breech is delivered, I think chloroform is preferable due to the quick and thorough anaesthesia it produces.

(4) The vagina is thoroughly ironed out with sterile green soap. In cases with a rigid pelvic floor. many obstetricians perform a deep episiotomy before commencing the delivery. Eardly Holland is very insistent on an episiotomy claiming that it relieves the head of the final quick compression that accompanies its exit which may cause lesions that are responsible for three-quarters of foetal mortality.

(5) Delivering the arms without entering the vagina. This ensures greater asepsis and may prevent delay in trying to reach an arm posteriorly. It also lessens the danger of deep lacerating, caused by putting a hand in a snug vagina alongside the baby's body.

(6) Failure of the head to engage may be caused by impingement on the symphysis. Pressure in the axis of the pelvis will only accentuate htis fault, while pressure directed towards the floor will correct it.

(7) When the mouth comes into view, the trachea is stroked with the fingers and mucus removed from the mouth, allowing the operator to deliver the head without haste.

(8) Potter protests against the habit of spanking or beating a baby as he claims it is unnecessary and may do harm. Nearly all babies will breath spontaneously if left alone and the heart is beating.

(9) Potter claims speed is not only dangerous but unnecessary in delivering a breech. He may take from eight to ten minutes after the umbilicus has come into view. This is confirmed by statistics quoted above showing the small number of babies that die of asphyxia.

In conclusion, I will remind you of the slogan posted in a prominent place in De Lee's delivery room which is particularly applicable to the handling of cases of breech presentation—"Non vis sed arte"—not strength but art.

"Too hasty delivery, and not asphyxia is the cause of the child's death in the majority of breech deliveries. The operator gets panicky when there is a slight delay in the progress of the extraction, and then makes violent, incoordinated and damaging motions, which leave their marks on the baby and also the mother. Gentleness, deliberation and a thorough knowledge of the mechanism of labor are the essentials for success."

Delusions.

By A. A. NUMBERS, 3T0.

Delusions are frequently seen in everyday life as well as in the clinic, in normals as well as in abnormals. Under these circumstances we should have some idea of how they arise, and when they should be considered as an indication of mental disease.

Definition—A delusion is defined as a false belief, the falsity of which is apparent, but out of which the person cannot be reasoned by indubitable evidence.

Many false beliefs have no pathological significance whatever. I may believe that to-day is Tuesday, when it is really Monday. That is a false belief while it lasts but it has only the significance of a mistake. We are all familiar with some rabid politician who can believe no wrong in his own party, the religious fanatic whose beliefs are untenable to so many, the man who lost his head over and makes so extravagant statements about "the one girl." The falsity of their beliefs seem quite apparent to us, but as they cannot be

reasoned out of them, we say they suffer from delusions. Yet in his day, Columbus' delusion was that the earth was round.

False beliefs, then, may be either normal or abnormal, and it is for us to endeavour to distinguish what constitutes a belief, first as false, and then, as such, what characterizes it as abnormal. According to one noted authority, there are certain main characteristics of pathological delusions in general:

1. They are as a rule very evidently not true to fact, highly improbable, even manifestly impossible often to the extent of being bizarre. Such for instances are the delusions of great wealth, of royal lineage, and of those of a certain class of patients who believe they have no stomach, no heart, even that they have no head.

2. They cannot be corrected by any appeal to reason; not originating in experience, they cannot be corrected by an appeal to experience. It is impossible to argue the patient out of his delusional beliefs.

3. They are out of harmony with the individuals education and surroundings. Should we find a Canadian with average education claiming that he could cast out devils, we would be justified in supposing him unbalanced.

4. It sometimes happens, however, that on the surface a false belief does not show any of these characteristics and yet may be pathological. A woman who says her husband is untrue to her has not voiced a belief which has on its face any evidences of impossibility, and with no knowledge of the facts, it cannot even be said to be improbable, and while not susceptible to the test by argument, it is certainty not out of harmony with the individual's education and surroundings. In such cases it becomes important to study the origin of the belief, to find out upon what sorts of foundation it is reared. If we find that it resulted from the patient's having awakened on several occasions during the night and found her husband's legs cold, and having reasoned from this that he had been out of bed to keep an oppointment with a paramour in an adjoining room, we will at once have no difficulty in stamping the delusion as pathological, because of being founded upon, and constructed of ideas which do no logically or reasonably lead to the conclusions reached.

As belief is the mental factor on which the delusion depends, it is necessary that we have some understanding of how beliefs arise in the apparently normal individual, in an effort to trace the mechanism of the disordered content of thought leading up to pathological belief.

Much has been written about belief being dependent on reason and judgment, and most people to-day cherish the idea that their beliefs are governed by these higher faculties. When the psychologists began to look into this, it was early recognised that belief is conditioned by desire, or at least holds a very definite relation to certain emotional constituents, and they are now bringing forward experimental evidence showing that our beliefs are in a large measure due to emotional and volitional determinations.

Lund, working under Prof. Hollingworth, and using group of students in two universities and a battery of propositions drew the following conclusions from his work:

1. Belief has a large emotional content. The correlation obtained between belief and desire was .88. The correlation between belief and knowledge .64, between belief and evidence .42, between knowledge and desire .03. The much lower correlations between belief and the more objective measures, support the inference to be made from the high belief-desire and co-efficients that, not only has belief a large emotional content, but emotional factors are significant determinants of belief.

2. There is a marked tendency to idealize the rational principle, and to conceive of its as the most valid and important of belief determinants, not-

withstanding the fact that non rational factors appear to outweigh it so largely in conditioning our belief attitudes. However, students, when rating themselves and others on a scale of rationality, do not consider rational factors nearly as important in conditioning the beliefs of their fellow students as in the case of themselves. They tend to rate themselves nearer the ideal than the typical individual.

3. Beliefs once formed, are not willingly relinquished, especially in the event of open commitment on a subject. Such unwillingness to give up one'e original position, is definitely related, if not responsible for the fact, that the side of the question first presented to us, and the first influences brought to bear upon us, are most effective in determining our beliefs, so much as to suggest the presence of a law of primacy in persuasion.

If it is true that in the normal, belief has a large emotional content and when once formed is not easily relinquished, the conditions are accentuated in the abnormal and much more inaccessible, so that it is difficult to trace the development of a pathological belief.

McDougall points out that false beliefs as to the estimation in which we are held by our fellow men, are very common among normal persons, we easily distort the significance of the remarks and attitudes of others. Not only do we falsely interpret indifferent remarks and attitudes, giving them a laudatory or depreciatory significance, but also are we apt to find such significance in actions or remarks that have no reference to ourselves. If we are aware of some weakness, or of some guilt, in ourselves, or if we feel that our merits have been insufficiently recognized, our sentiments of self-regard becomes unduly sensitized; its desires then play an undue part in our lives. In the former case (weakness or guilt) we shrink unduly before the regard of our fellows and tend to interpret them as depreciatory. In the latter case (merits insufficiently recognized) we constantly resent the indifference of others to our merits and strive in imagination, in fantasy, or in action, to assert ourselves, to display our superiority, or at least our worth and capacities. And commonly thes two tendencies of our self regard operate alternately or in conjunction; producing that state of conflicting self assertion and submission which we call embarrassment. Few men, however soundly constituted, pass through life without suffering some such conflicts, and without entertaining, however fleetingly about their personal relations, false beliefs, springing from these impulses of self regard.

Such delusions are morbid as soon as they become so fixed that they cannot be rectified by cool reflection and in the light of further evidence, such as assurances or respectful treatment accorded us by our friends and acquaintances. Often such mild delusions are expressed in such forms as "luck is always against us" or "Fates are against us" or "An honest man does not get his due in this world." A rather more dangerous and more definite degree of the delusion takes the form "Everyone seems to be against me," or "Nobody cares about me; they are all down on me."

It is probably only when the subject is aware of some shameful weakness or of some guilty action in his own past, and when he represses in some degree this knowledge and its affective tendencies, that such delusions become so fixed as to fall into the distinctly borbid class. The repression removes them from the reach of frank self-criticism, and prevents the expression of the tendencies in natural channels of speech and action. Hence the introvert is more liable to the development of delusions than the extrovert; for the former hugs his resentment and his desire for recognition in his own bosom; while the latter at once gives vent to his injured feelings in loud complaints or to his self assertion

in boasting, or in open efforts to display his powers and to compel recognition from his peers.

While there is, no doubt, a great many variations in the way pathological beliefs develop, the following would appear to be one of the most probable and common:

1. Marked emotional upset.
2. Effort to rationalize out of a pain- situation.
3. Conflict and repression or failure to compensate, leading to undue mental sensitization and transference, going on to the development of suspicions.
4. Systematization of suspicions, apparent as delusions of persecution.
5. Going a step further and rationalizing for persecutions leading to delusions of grandeur.

With the sensitization and transference, the emotional state or affective tendency plays an essential part, usually through psychic pain, distorting observation and reasoning, or facilitating acceptance of some false proposition, which then seems to be preserved in an airtight compartment secure from the disturbing influence of hard fact and indubitable evidence, and backed now by primacy in persuasion. The delusions are, under these conditions, highly disguised with formations, substitution products, compromise formations; or they may be defense reactions, avoidances, compensations, or projections. They differ in the obviousness of their meaning to the extent that the ego has deteriorated.

Freud and his school think that here, as well as in other mental conditions, the emotional state giving rise to the delusion may be traced back to sex difficulty, particularly impotence and homosexuality and that careful psychoanalysis may reveal this, and enable some attempt at treatment.

Classification—Owing to the variety of delusions, the large number of mental conditions in which they are found, the obscurity of their cause, and inaccessibility to investigation, no uniform classification has yet been agreed on. Rosanoff divides them into three great categories:

1. Melancholy ideas.
2. Ideas of persecution.
3. Ideas of grandeur.

1. Melancholy ideas have several varieties.
   (a) Ideas of humility, culpability, self-accusation.
   (b) Ideas of ruin.
   (c) Hypochondriacal ideas.
   (d) Ideas of negation.
2. Ideas of persecution are divided into two classes:
   (a) Accompanied by hallucinations.
   (b) Unaccompanied by hallucinations.
3. Ideas of grandeur:
   (a) In acute psychosis.
   (b) Without deterioration.
   (c) With deterioration.

White's classification is altogether different, his groupings being:

1. Whether they relate to the outside world, including outside persons.
2. Whether they relate to the individual's own personality.
3. Whether they relate to the individual's own body, and may be
   (a) Fixed or changeable,
   (b) Non systematized or systematized,
   (c) Endogenous or exogenous.

It is frequently stated that as delusions are found in so many forms of mental disorder, they can be considered only as symptoms, the mere superficial indications of a deep seated and widespread disorder. I think we can all agree that it is a symptom, probably caused by conflict and psychic pain, but that it is indicative of deep-seated and widespread disease is open to question since we find in paranoia that the delusion is the essential and sole symptom, accompanied by the complete retention of clearness and order in thinking, willing and acting, unless the delusional system is touched on.

The delusional system, however, is but the far advanced stage of a long

chronic process, the cause of which is seldom found. One writer likens the delusion to scar tissue, the result of an old wound, the cause of which can only be guessed at.

Treatment: What then can be done in the way of treatment? Where there is mental deterioration little or nothing can be done. With no deterioration, but marked systematization, removal from environment and suggestion may give some results, but most cases would appear to be hopeless since the cause cannot be determined or removed. The hope is that mental hygienic education at puberty will do much to warn and protect the individual through early recognition of a situation tending to develop delusional formations.

SUMMARY:

Delusions are pathological beliefs
1—evidently not true to fact,
2—cannot be corrected by any appeal to reason,
3—out of harmony with the individual's education and surroundings,
4—or in some casese due to false interpretation of observed facts.

Such beliefs have behind them an accentuation of the factors governing normal processes.

There are various mechanisms of development usually starting out from emotional upset, conflict and repression.

Regarding personality types involved delusions are more apt to arise in introverts than in extroverts.

Present classifications are not satisfactory.

Delusions are only symptoms of mental disorder with some cause behind them.

Treatment is preventive through early education.

Arterio-Venous Aneurysm.

By J. H. FORRESTER, 3T0.

To William Hunter, in 1757, properly belongs the credit of first describing accurately the clinical features and the local and regional vascular disturbances of acquired arterio venous aneurism known, when Sir Prescott Hewitt reported his case. Since then many cases have been reported, but most of our knowledge has been accumulated on this subject, in the last 15 years and is derived from the extensive work of Reid, Holman, Halsted and Matas of this continent, and Makins, Drury and Lewis of England.

Arterio-venous aneurism is defined as being any abnormal communication existing between an arterial and venous channel, but is usually limited only to communications between the larger vessels. This, in spite of Reid's, who takes a greater scope, also calling circoid aneurisms and simple angiomata of the peripheral vessels, arterio-venous aneurisms.

Etiology and Pathogenesis.

Etiologically, A. V. Aneurisms may be classified as A—Acquired and B—Congenital.

A.—The acquired are subclassified thus—

1. Traumatic—Callander surveyed 447 cases of A.V. aneurism and found that 85.7% bore a definite relation to trauma. It is of interest to not the common occurence of arterio-venous fistula at the time that venesection was practiced so assiduously. Hunter's two cases had such a traumatic origin. Trauma is usually in the form of small puncture wounds such as occur from gunshot wounds or secondly, from knife stabs. Less commonly, these aneurisms may follow a severe contusion, and

very occasionally, as a sequal to a fracture in which the vessels are lacerated.

2. Infection—This is a rare etiological factor. A fistula may arise when an abcess, involves the walls of an opposing artery and vein such that the former ruptures into the latter. Cases have been reported in which infection in an amputation stump has resulted in a similar rupture of the ligated artery and vein.

3. Spontaneous or Idiopathic—41 or 10% of Callander's cases presented no etiological factor. In the opinion of the writer that some of these cases were of congenital origin, their clinical manifestations being submerged in early life.

4. Secondary A.V. aneurism—These arise secondarily to true arterial aneurisms. They are very rare, usually occurring near the heart, and result from the gradual erosion of the vessel walls until rupture of the artery and vein occurs.

B. Congenital A.V. aneurisms are comparatively rare. Callander found only three or less than 1% in his survey. These aneurisms have usually multiple fistulae, and occur in the head, neck, and extremities respectively. An insight into the factor of their production is found in the work of Sabin & Woolard on the embryonic vascular system. They found that the direction of flow in certain embryological vessels was reversed at different stages of development. They point out that arteries and veins arise by a differentiating process from the same capillary plexus. When the subclavian vessels are beginning to differentiate, multiple communications have been observed between the artery and vein. So on this basis, it is assumed that all congenital A.V. aneurisms result from anomalies of vascular development. It is of interest that often these cases are associated with anomalies of other systems, particularly hydrocephalus.

The **Incidence** of A.V. aneurism is intimately associated with warfare due to the use of small bore bullets and shrapnel. The incidence in civil life is much lower although not so much as is generally held. Four cases were treated in H.S.C., Toronto, in the past year.

The **site** of these lesions in the body is regulated largely by the degree of opposition of large arteries and veins in certain positions, and the chance of these positions being traumatized. The commonest sites are, the popliteal space, the femoral sheath, the antecubital fossa, the carotid sheath. In the cranium the fistula is almost invariably between the sinus and the internal carotid artery.

The **origin** of A.V. aneurism, then, is dependent in nearly all cases to a simultaneous trauma to the walls of an opposing artery and vein. The fistula may arise at once or if the walls are merely weakened, may not arise till the arterial pressure completes the rupture in a few days.

Anatomically, A.V. aneurisms are classified thus: A.—Aneurysmal Varix —the arterial and venous walls are directly adherent about the fistula. It is the common type which only occurs when the involved vessels are normally in close opposition, and the trauma such that little perivascular hematoma forms, tender such conditions, early agglutination of the torn edges of the apertures is possible.

B—Varicose Aneurism. Here, the above conditions being reversed, a pulsating hematoma forms in the perivascular tissues. This in time becomes encysted and lined by endothelium, forming a sacculous communication from vessel to vessel.

Many sub-types arise secondarily from those general types, depending on the site of the lesion, the peculiar trauma and the chronicity of the fistula.

The Pathology of arterio-venous aneurism is best considered under 3 heads—

A.—Local effects,
B.—Regional effects,
C.—Systemic effects.

A.—Local effects,—1. In the vein, both proximal and distal to the fistula there is a dilation. This is due to the increased venous pressure. The degree of dilation depends primarily on the amount of arterial blood passing through the fistula and secondarily, on the resistance of the perivascular tissues. Distally this dilitation extends as far as the first functioning vessel while proximally it varies inversely as the distance from the fistula. The muscular coat, and to some extent the elastic coat of the vein, undergo hypertrophy and hyperplasia. This is called arterialization of veins and is unique evidence of the adaptation of a tissue to functional demand.

2. In the artery, local changes are diverse and the cause of their production is not fully understood. The artery distal to the fistula may be small and contracted, normal, or dilated with evidence of degeneration of its wall. Holman gives a plausible explanation for this. He claims that primarily the vessel is narrowed because it is receiving a reduced volume of blood under reduced pressure. In long-standing cases, an abundant collateral circulation to the distal parts is formed and forms large anastomosis with the distal artery—when this occurs, Holman believes blood passes in a reverse direction in the artery back to the fistula. When this reversed flow attains a large volume, the artery compensates by a dilatation.

The artery proximal to the fistula is invariably dilated and tortuous and its walls are thin, less elastic and less resistant. The muscular elements show atrophy. Two theories have been evolved to explain this. Holman, of Boston, claims this is a compensatory dilatation. He states that the local, lowered peripheral resistance created by the fistula attracts blood to this new heart-artery-fistula-vein circulation rather than to the normal heart-artery-capillary-vein circulation. The artery dilates to compensate for the increased flow. Reid, of Johns Hopkins, claims the above theory to be untenable; that Holman has disregarded the degenerative changes in the vessel wall; and that if it were purely a question of handling an increased blood volume, one would expect to see an hypertrophy of the wall, which is not the case. Reid believes these changes in the proximal artery can be explained by Thomas idea, that a normal diastolic pressure is essential to the integrity of any vessel wall. Such may be true; however one would expect general effects in all arteries as the low arterial pressure is a general effect. Of these two theories, Holman's is generally accepted.

These local vessel changes bear a definite relation to the size of the fistula and its chronicity. If it is small, there may be very little change. If the fistula is larger than the artery itself, the only curb to an increasing blood flow at first would be the distensibility of the artery and the resistance of the perivascular tissues. This dilatation would increase due to an increased minute blood flow until the resistance of the fistula, plus the resistance of the vessel wall and the perivascular tissues equals the resistance of the capillary bed.

B. — Regional Effects, — These, of course, depend on the site of the lesion. The temperature in the region of the fistula is 1-2° F. above normal due to the abundant collateral circulation is offset by stasis due to the unbalanced circulation. Reid believes the presence of a fistula constitutes a powerful stimulus to the establishment of a collateral circulation and has proved this experimentally. While no one can dispute the fact that an abundant collateral circulation does form, no one has yet offered a tenable mechanical basis by which it might occur. A test for collateral circulation in these cases, is to compress the fistula and observe the rapid return of the pulse in the distal arteries.

Increased girth of the limb distal to the fistula occurs. This is due to

the the increased vascularity of the part as a result of stasis, and to an hypertrophy of fat and connective tissue. If the epiphyses have not united, marked lengthening of the limb occurs due to excessive bone growth. The hair may be excessive on the part.

Trophic changes also occur distally. These are attributed to (1) diminished flow to peripheral capillaries; (2) stasis of the reduced blood as a result of increased venous pressure. The resulting tissue anoxaemia causes progressive effects such as muscle atrophy and pigmented brawny skin which tends to ulteration. Cyanosis is common. The regional superficial veins are varicosed The arterial pulse in the periphery is diminished or lost and the blood pressure here is less than in the other limb.

C.—Systemic Effects—When the fistula is created, certain disturbances of the circulatory balance occur. The mean blood pressure is lower; the size of the vascular bed is increased by local dilatation of vessels and engorgement of parts distal to the fistula; a considerable volume of arterial blood is being shunted direct into the venous system. To compensate for the above, there is a progressive increase in the total blood volume.

Cardiac enlargement, as a result of hypertrophy and dilatation is present in many cases. In view of the increased venous pressure, American authors believe this is a compensatory mechanism; that the dilatation takes care of the increased venous return while the hypertrophy is a response to the demand for greater minute output. There is a faster pulse rate. This is in accord with Krogh's theory that given adequate venous filling the cardiac output is increased by a faster contraction rate. English workers, Lewis and Drury, failing to find any increased venous pressure or cardiac output claim the cardiac enlargement is due only to dilatation as a result of deficient coronary circulation. That is, they assume a nutritional disturbance of the heart due to the low mean blood pressure. This is very well taken, but most experimenters have invariably found increased venous pressure associated with an increased cardiac output, even as high as 100%.

The systolic blood pressure falls but little compared to the fall in diastolic pressure due to the fault being a lowered peripheral resistance. This results in a high pulse pressure and accounts for the capillary pulsation and the water hammer pulse found clinically. The pulse rate is increased 10-30 beats.

It is interesting at this point to consider at this point the immediate and remote effects of closure of the fistula.

Immediate Effects:—

1—A rise in the general blood pressure to a high point for a few minutes, with a gradual fall to a level above that present before the fistula was produced. This involves both systolic and diastolic pressures, particularly the latter.
2—An immediate and marked bradycardia (Branhams sign).
3—Decreased venous pressure proximal to the fistula.
4—The return of the arterial pulse in the distal part.

The remote effects, occuring in the next 2-3 months are the gradual return to normal of the blood volume pulse rate, blood pressure and any regional disturbances.

Symptomatology and Diagnosis:

The diagnosis of A.V. aneurism is usually easy. The three cardinal findings are:—

1—Thrill,
2—Murmur—These are heard and felt respectively over the fistula. There, they have their P.M.I. and both are propagated widely along the vessels. They are continuous and have systolic exacerbations.
3—Tumor mass which presents an expansile pulsation; associated with these local signs, are, fast pulse high pulse pressure, water hammer and

capillary pulse. It is of interest to note the similarity of these findings to those of aortic regurgitation. Later signs such as the cardiac enlargement and regional changes have been discussed under pathology.

The cardinal signs plus the history and local evidence of trauma usually suffice to make a diagnosis. True arterial aneurism is sometimes confused. In the latter, the thrill and murmur are limited to systole alone. In cases of doubt, the recovery of arterial blood from the proximal vein is pathognomonic.

Prognosis:

The prognosis must be guarded in the first few days as death of the patient or gangrene of a part may result from hemorrhage or infection.

In the event of an uncomplicated early period, the aneurism takes a chronic course. Cases have persisted 40 years without marked disability. Fistulae occuring in the head and neck region are best tolerated. Untreated fistulae undoubtedly shorten life by producing serious cardiac involvement while locally gangrene or ulceration are always potential dangers. Spontaneous cure does occur but only in the first six months after production of the fistula. These heal by a restorative process.

Treatment:

Operative treatment is best for arteriovenous aneurism. Treatment by compression, by injection of coagulating fluids and by galvanopuncture have been discarded from modern procedures as not only useless but dangerous.

The time to operate is an important consideration. In such cases as severe hemorrhage, increasing hematomata or infection, there is no choice; immediate operation is imperative. Apart from these early complicated cases, the operator may choose his own time. During the Great War, surgeons of the allied armies postponed operation 3 to 4 months while the Germans operated within 3 weeks of the injury. The present concept, evolved by Reid and Makins, is that operation should be postponed until 6 months after the injury, during which time rest in bed with immobilization of the part should be practiced. The advantages of such a procedure are—

1—A good collateral circulation becomes established.
2—The wound is well healed.
3—The perivascular tissues are free of hematoma, thus decreasing the danger of infection.
4—The patient is a better operative risk as all signs of shock and initial haemorrhages are gone.
5—The fistula may heal spontaneously.

Types of Operation:

Ligation of the proximal artery alone or ligation of both proximal and distal arteries have been condemned on the grounds that they not only fail to cure the aneurism but also that these operations are often followed by gangrene of the distal parts.

Two operations designed to restore the vascular channels are still used. The first is ligation of the canal of communication. The second is a lateral or end to end anastomosis of the vessels. These two procedures yield very good results but as well as being difficult, there is much doubt of the advisability of restoring a damaged vessel to normal function. Indeed, true arterial aneurisms have occured in these restored arteries.

The operation of choice is complete extirpation of the aneurism. This entails quadruple ligation of the main vessels plus ligation of any small vessels communicating with the aneurismal sac. Some operators stop at this stage while others completely remove the aneurism by incising within the above ligatures. This operation can be done on all cases, gives excellent results and is easier to perform than the above restorative operations.

In Summary:

Arteriovenous aneurism is any abnormal communication between an artery and vein. They are largely acquired lesions, and some form of trauma is the etiological factor of their production. The incidence is rare except during warfare. The effects of the fistula may be summed up under —Local—in which the vein dilates and is arterialized by increased pressure and the proximal artery atrophies and dilates to compensate for increased blood volume. Regional effects—an increased local temperature and decreased temp. distally; hypertrophy of bone and subcutaneous tissue with atrophy of muscle and skin; well developed collateral circulation. Systemic effects—increased blood volume and cardiac dilatation and hypertrophy occuring as compensatory mechanisms. Rapid filling of the heart leads to increased output due to faster rate. The diagnosis is easy, and based on a history of trauma associated with continuous thrill and murmur at the site of the fistula. These are pathonomognic if they are continuous with systolic exacerbations and are lost on compression of the fistula. The prognosis of treated cases is very good. The treatment is surgical. Six months of conservative observation followed by operation in which the entire aneurism is extirpated is recommended.

"Among Giants."

By MIRIAM A. BRICK, 3T0.

According to the ancients, the earliest inhabitants of the earth were a race of giants. These superhuman men of monstrous size and strength were believed to personify the elements of Nature, and legend tells how they sprung from the blood of Uranus (Heaven), which fell into the lap of Ge (the Earth). A still later conception, recognized by Pliny in the early part of the Christian era and adopted as a doctrine in the theology of Augustine, was that the humans had degenerated in shape and size from a primeval race of Giants. The cyclops of Ulysses' voyage have been made immortal by Homer. Dating from the earliest inhabitation of England there is a legend relating how the brother giants Gog and Magog came to stand in the gallery of the Guildhall in London that is closely linked with the history of the early Britons.

Up to the end of the nineteenth century giants are of antiquarian interest only. Among the anatomical curiosities displayed in Paris during 1867 was a Chinese giant Chang Wu Gow, who stood seven feet nine inches. In 1905 Wackon, a Russian, was exhibited in London. He was then twenty-three years old and nine feet three inches high. Frederick the Great is reported to have had a gigantic Scotsman, eight feet seven inches in height, in his personal bodyguard. Curiously, these creatures were usually of feeble intelligence, languid disposition and rarely lived beyond middle life.

A conspicuous specimen in the College of Surgeons, London, is the skeleton of a man seven feet seven inches high. This skeleton's claim to fame rests on its association with John Hunter, surgeon and anatomist. The story of its acquisition is characteristic of John Hunter's enthusiasm in procuring subjects for his museum or biological investigations.

At the time John Hunter was making his investigations in comparative anatomy there lived in London a gigantic Irishman, O'Brien by name. Hunter was acquainted with the giant and had long hoped to add his skeleton to his

interesting collection. When, in June, 1783, O'Brien's health began to decline, Dr. Hunter sought permission to dissect the body at the giant's death. The Irishman was horrified at the idea of being dissected and refused to make any such compact. Instead he took all precautions lest his corpse should fall into the hands of Hunter. He charged certain of his countrymen that following his death they should safely guard his body until a leaden coffin could be made in which it would be conveyed to the sea for burial.

Failing in his request Hunter sent his servant Hewison to stalk all O'Brien's movements that he might be certain of obtaining his corpse. When the giant died Dr. Hunter interviewed the guardians of the body in an alehouse and offered them £50 if they would permit him to kidnap the body. The men recognized the scientist's eagerness and raised their demands to £500. Hunter borrowed the money, paid off the guards, and, entering the barn in which the coffin had been hidden, substituted heavy stones for the body. The prize was then conveyed with all speed by hackney and his own coach to his laboratory at Earl's Court. Lest it should be recognized by his friends, Hunter had the skeleton immediately prepared for his museum. This expensive specimen now forms a part of a collection estimated at 13,682 subjects accumulated by Dr. Hunter which were later purchased by the government and are now in the College of Surgeons, London.

Acromegaly was first described with exactitude by Pierre Marie in 1886. Marie related five of his own cases and reviewed the literature on similar histories. Following the recognition of acromegaly as a well-defined disease which is frequently associated with giantism, renewed attention was directed to its investigation. During the past thirty years more scientific research has explained the remarkable and gigantic individuals seen in the side-show on the basis of hyperendocrine secretion.

Sternberg in 1898 classified gigantism into normal and pathological giants. The former are normal individuals in good health, who are exceptionally tall and live to old age. Of the abnormal giants he claims forty per cent demonstrate gigantism with acromegaly. The remainder is made up of gigantism associated with leontiasis ossium, Paget's osteitis deformans or following congenital lues, tumors of the testes, castration.

In 1906 Lannois and Ray, and in 1911 Cushing published the results of their investigations on gigantism. They pointed out that most cases of human giants were due to hypersecretion in anterior lobes of pituitary body, occurring during growth period, that is, prior to epiphysial ossification.

"A doctor sent me his son to be taught such verses as these,
'Sing the wrath and many the woes that were sent.'
He learned them, but when we came to the following sentence:
'Many valiant souls he sent down headlong to Hades,'
He sent him no longer to me as pupil, and when
He saw me, 'Thank you,' he said, 'my friend, but my son is quite able
To learn these things from me at home, for I also
Send many valiant souls down headlong to Hades,
And am not in need of a teacher to show me how.'"

—From Lucullus.

Some References to Ancient Greek Physicians

"Marcus the doctor called yesterday on the marble Zeus; though marble, and though Zeus, his funeral is to-day."

"Diophantus, the astrologer, said that Hermogenes, the physician, had only nine months to live; and he laughingly replied, 'What Cronus may bring to pass in nine months do you consider; but I can make short work with you.' He spoke, and reaching out, just touched him, and Diophantus, while forbidding another to hope, gasped out his own life."

"Diophantus, having seen Hermogenes, the physician, in sleep, never awoke again, though he wore an amulet."

On an oculist:

"If you have an enemy, Dionysius, call not down upon him Isis nor Harpocrates, nor whatever God strikes men blind, but Simon; and you will know what God and Simon can do."

"Agelaus killed Acestorides by an operation; for, 'Poor man,' he said, 'he would have been lame for life.'"

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